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Paraquat Poisoning

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report of a series of 16 cases of Hodgkin's disease involving Waldeyer's ring.1 In our series there was involvement of the nasopharynx in eight cases but, unlike the Swedish report, our cases were all ones in which biopsy of the nasopharynx was indicated by symptoms related to this area. In two of our patients the disease was primary in the nasopharynx; the other six had more widespread disease at the time of nasopharyngeal involvement. Seven of the eight cases were of the mixed cellularity type, the other showing a lymphocytic predominance type of change. None of our nasopharyngeal cases showed the epithelioid granuloma type of Hodgkin's change first described by Lennert and Mestdagh,² in contrast to our seven tonsillar cases, four of which showed this change; this observation suggests that tonsillar Hodgkin's disease may often be a separate form of the disease. The cervical nodes that were involved in these cases also showed this epithelioid appearance.

Although in our series the course of the disease process did not seem to differ from that of the general pattern of Hodgkin's disease, it is possible that the presence or absence of microscopical Hodgkin's disease in the nasopharynx of a patient with disease elsewhere might be clinically important. The usefulness of this investigation could be ascertained only by carrying out a series of biopsies of the nasopharynx on an unselected group of patients with Hodgkin's disease at the time of primary diagnosis and comparing the findings with those in other tissues and with the fate of the patient. A detailed report of the findings of Dr Biörklund and his colleagues will therefore be awaited with interest.

Most cases of Hodgkin's disease present with enlarged lymph nodes. It is reasonable to postulate that the disease originates in Waldever's ring (an earlier station of the lymph node stream draining into the cervical lymph nodes) whether it spreads as a neoplasm or as a virus. The nasopharyngeal findings of Dr Biörklund and his colleagues and of ourselves are compatible with this concept and suggest that histological study of the nasopharynx in Hodgkin's disease will also further our fundamental knowledge of the disease process.

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- ¹ Todd. G B, and Michaels, L, Cancer, 1974, 34, 1769.
- ² Lennert, K, and Mestdagh, J, Virchows Archiv (Pathologische Anatomie), 1960, 344, 1.

Paraquat poisoning

SIR,-Fortunately the incidence of accidental paraquat poisoning in the United Kingdom seems to have receded, but the number of cases of deliberate self-poisoning with this chemical persists at a worrying level. Many of the people concerned are apparently unaware of the ultimate consequence of their acts.

An indication to prognosis can be derived from a relatively simple test on the urine. This test relies on the reduction of paraquat to a blue free radical by alkaline sodium dithionite. The reagent must be freshly pre-

pared by adding 10 ml of 1M sodium hydroxide (1 mol/l) to 100 mg of pure sodium dithionite. Aqueous paraquat standards are prepared containing 1.0, 5.0, and 10.0 mg/l (µg/ml) of paraquat ion and 2-ml aliquots of the sodium dithionite reagent are added to 10-ml volumes of the test samples and paraquat standards. Quantitation is by visual inspection and the test is sensitive to 1.0 mg/l (μ g/ml) in clear urine. Since diquat is reduced to a green free radical, a green to blue colouration indicates Weedol ingestion.

If no colour change occurs (that is, the result is negative) it can be assumed that no significant quantity of paraquat is being excreted and accordingly that no toxic amount has been absorbed. Active treatment can then be withheld. On the other hand a colour change to some shade of green or blue, while being no absolute criterion, certainly points towards an unfavourable outcome. These people, we think, should definitely be candidates for treatment. The first purpose of this letter, therefore, is to urge the provision in all emergency departments of facilities for this test throughout the 24 hours.

Regrettably at this stage no form of treatment can be confidently recommended, although several possibilities are being actively pursued at various centres throughout the country, the immediate treatment involving prompt but careful gastric aspiration and lavage, after which 500 ml of a 30% suspension of fuller's earth BP together with 5% magnesium sulphate is left in the stomach. The intention is to minimise absorption in the manner demonstrated in the successful findings of experiments in rats.1 Further details on this and the continuing treatment are available from this unit.

Animal experiments have also demonstrated that charcoal haemoperfusion produces both a good clearance of paraquat from the blood² and survival.³ In this unit, as well as in several others, we are now studying the effectiveness of that technique in patients. For this purpose we need to receive patients with the least possible delay following ingestion and the second purpose of this letter is to invite clinicians who are confronted with cases of this kind to telephone us at once with a view to their transfer (01-407 7600, ext Poisons).

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- 1 Smith, L L, et al, British Medical Journal, 1974,
- 4, 569.
 Maini, R, and Winchester, J F, British Medical Journal, 1975, 3, 261.
 Widdop, B, Proceedings of Conference of the European Society for Artificial Organs, Berlin, 1975, to be published.

Effect of smoking on carboxyhaemoglobin level in pregnancy

-Mr T G B Dow and his colleagues (1 November, p 253) report that there is a significantly greater rise in blood carboxyhaemoglobin (COHb) concentration in response to smoking a single cigarette in pregnant as opposed to non-pregnant women.

However, their study was confined to the second trimester of pregnancy and the subjects were instructed to puff every 40 seconds, inhaling as deeply as possible to a total of 10 puffs per cigarette.

I would like to point out that a different picture is obtained if subjects are allowed to smoke naturally. This approach was adopted in a longitudinal study of 10 pregnant smokers who were followed at 4-8-weekly intervals from 12-16 weeks of pregnancy to six weeks postnatally. The subjects were aged 19-30 years and had smoked 5-30 cigarettes daily for at least five years. At each attendance the subjects smoked one standard tipped cigarette (nicotine delivery 2·1 mg) in their "normal" manner. All tests were conducted at 9 am and the subjects had not smoked for 1½ hours before smoking the test cigarette. Details of subjects and procedure are described elsewhere. The number of puffs per cigarette decreased as pregnancy progressed from a mean of 14.66 ± 0.76 puffs per cigarette at 12-16 weeks (six subjects) to 11.4 ± 0.93 at 33-40 weeks (10 subjects). Postnatally, the number of puffs per cigarette rose to 12.9 \pm 0.98 (10 subjects). However, in the same subjects there was no significant change in the nicotine obtained from the cigarette at different stages of pregnancy.

In some of these subjects (not always the same subjects at each stage of pregnancy) estimations of blood COHb were obtained immediately before and immediately after smoking. Blood was taken from finger-prick samples (after preliminary experiments which showed that finger-prick and venous blood samples gave identical results) and COHb was estimated by the method of Commins and Lawther.2 The results from these few estimations showed that, like the puffing rate, the rise in COHb concentration after smoking decreased as pregnancy progressed, from $3.17 \pm 0.20\%$ at 12-16 weeks to $1.09 \pm 0.31\%$ at 33-40 weeks. Six weeks postnatally there was an increase to $1.80 \pm 0.34\%$. changes in COHb while smoking are shown in the figure, along with the puffing and nicotine data from the same subjects.

These observations indicate that women alter their smoking behaviour during pregnancy. Other observations have shown changes in smoking behaviour during stress and relaxation3 and with different strengths

